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Response of Associated Oral Soft Tissues When Exposed to Argon Laser During Polymerization of Dental Resins

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Background and Objective: **Polymerization of dental resins with Argon laser produces restorations with improved physical properties when compared to conventional visible-light polymerization techniques. However, the possibility of damaging adjacent soft tissues has not been addressed.**
Study Design/Materials and Methods: **In this study, Argon laser (488/514 nm) was used for the polymerization of composite resins to determine effects on the parakeratinized gingiva adjacent to both restored and unrestored teeth in six dogs, using 10-, 20-, and 30-second polymerization exposures.**
Results: **Gingival tissues removed at 24 hours, 72 hours, or 5 days revealed desiccated, disrupted, hyalinized connective tissue. Tissues exposed for 10 seconds showed minimal change. This minimal degree of change was most evident at 72 hours and returned to normal limits at 5 days. The 20-second exposure produced alterations evident through all time periods. Tissues exposed for 30 seconds exhibited necrosis, severe disruption, and vessiculation, which was still unresolved at 5 days.**
Conclusion: **This study demonstrates that clinically relevant Argon laser exposure (10 seconds) of parakeratinized gingiva adjacent to teeth undergoing restoration does not cause lasting damage. Lasers Surg. Med. 20:467–472, 1997.**

Key words: Polymerization of dental resins; Argon laser tissue thermal effect; Argon laser wound healing; Argon laser exposure times

INTRODUCTION

Studies have demonstrated that polymerization of photo-activated dental resins with the Argon laser produces a resin with superior physical properties [1]. Although studies have shown that this system produced no adverse effects on the dental pulp [2], the possibility of laser damage to the adjacent gingiva has not been investigated.

Many authors [3–6] have discussed the tissue reactions of Argon laser treatment of the skin, especially involving the removal of tattoos, but there is a paucity of information regarding Argon laser effects on the intraoral parakeratinized mucosa. The dental literature, which contains numerous reports on the effects of CO_2 and Nd:yag lasers on the periodontal tissues, has not reported much information on Argon laser tissue effects. Only, Skink et al. [7] have discussed Argon laser effects on a nonorthokeratinized epithelium, the simple squamous epithelium of the peritoneum.

This study is designed to identify and describe possible damage to the parakeratinized gingival tissues adjacent to laser-treated restorations, at power settings appropriate for polymerization of resins and using direct Argon laser exposure of the tissues at three different time settings.

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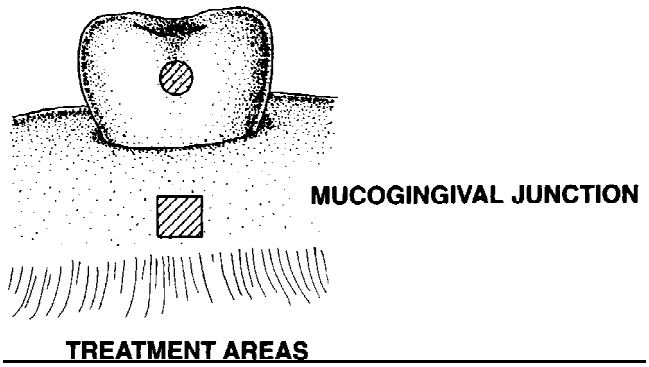

Fig. 1. Treatment areas: circle—restoration preparation; square—lased soft tissue.

MATERIALS AND METHODS

Subjects

The experimental animals used in this study were six adult dogs of indeterminate breed. There were three females and three males, each weighing ~25 kg. Several of the animals exhibited variable amounts of gingival pigmentation. Visual examination revealed no overt carious lesions and only minimal amounts of cervical deposits (calculus).

Placement of Restorations

The animals were sedated with ketamine/AcePromazine/Atropine in a 10-1-1 dose (30mg/kg IM). Anesthesia was induced using Thiamylal Sodium (Bio-tal) 2% solution injected IV.

Standard one-surface composite restorations were placed on the buccal surface of four teeth in the mandibular right quadrant (Fig. 1). Each restoration was cut with a #34 inverted cone burr in a water-cooled highspeed handpiece. Depth of preparation extended past the dento-enamel junction uniformly. Restorative material (Prisma) was placed in each restoration using standard acid-etch technique.

Curing of Restorations

Three of the prepared restorations were polymerized with Argon laser (488/514 mn) operating at 231 milliwatts with a 5 mm spot size, for either 10 seconds, 20 seconds, or 30 seconds. As part of each individual restorative procedure, a bonding agent was placed in each restoration and Argon laser polymerized with an additional 5-second exposure. Thus each tooth received either 15 seconds, 25 seconds, or 35 seconds of total Argon laser exposure. The fourth restoration was polymerized for 40 seconds (the usual exposure also using a 10-second exposure for the bonding agent) with Visible Light (Caulk Max-light). Treatment was completed on all animals at the same session with the same operator performing all restorative procedures.

Soft Tissue Treatment

As the restorations were being placed, there was concurrent exposure of the mucogingival junction buccal to each tooth at the exposure level of the finished restoration—either 10 seconds, 20 seconds, or 30 seconds of Argon laser, or 40 seconds of Visible light (Fig. 1). In addition, the same *unrestored* teeth in the left mandibular quadrant and their adjacent mucogingival junctions were treated with identical amounts of either Argon laser or visible light. This provided six separate laser exposure areas and two similar areas of soft tissue exposure for each animal.

Controls

Several control tissues were obtained including: (1) Visible Light (VL) exposure adjacent to a tooth with a similar restoration, (2) VL exposure to gingiva adjacent to an unrestored tooth, (3) untreated tissue adjacent to an unrestored tooth. Tissues were examined at 24 hours to assess the initial reactions, at 72 hours to determine the intermediate reaction, and at 5 days to determine the potential for permanent damage.

Tissue Preparation

Two animals (#3820 and #3930) were sacrificed at 24 hours with Beauthanasia D. Mucogingival soft tissues were removed with scalpels down to the periosteum. Buccal attached gingiva was freed with a horizontal incision around the cervical portion of the teeth. A second horizontal incision was made subjacent to the mucogingival junction. Vertical incisions were then made separating the tissues into sections corresponding to the associated teeth. A circumference of normal, unaffected tissue was included in each specimen. The individual samples were dissected free of underlying bone and placed in 10% formalin. There were nine tissue samples for each animal.

Each tissue sample was sectioned vertically from free gingiva to movable mucosa through the center of the treated area, thus providing progressive sections. Histologic sections were prepared using Hematoxylin and Eosin stain. Two additional animals (#3823 and #3826) were sacrificed at 72 hours and the final two animals (#3825 and #3836) at 5 days. Tissues were removed and slides prepared from all animals in a similar manner.

TABLE 1. Controls*

Tooth #	Restored	Cure	# of tissues	Changes noted
$R\text{-}P_2$	Yes	VLC-40 sec	6	0
$R\text{-}P_1$	No	VLC-40 sec	6	0
$L\text{-}P_2$	No	No	6	1

*Seventeen of the 18 control tissues showed no abnormal alterations.

RESULTS

Controls

No soft tissue changes were noted in the 18 histologic sections associated with either the untreated controls or the Visible Light cure controls with one exception. At 5 days, one untreated control exhibited chronic inflammation along the epithelial-connective tissue junction and slight disruption of the connective tissue that may represent an area of injury or gingivitis (Table 1).

Microscopic examination of the other control soft tissues revealed normal epithelial progression from nonkeratinized crevicular epithelium to marginal epithelium to attached parakeratinized epithelium to parakeratinized movable mucosa. The underlying connective tissue contained chronic inflammatory infiltrates that were most abundant in the crevicular area and scant elsewhere, as expected (Fig. 2).

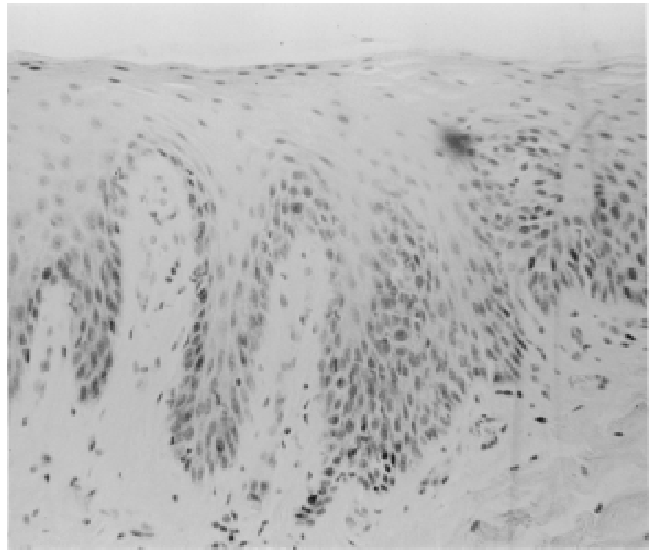

Fig. 2. Photomicrograph (Hematoxylin and Eosin Stain × 100) of normal parakeratinized epithelium at the mucogingival junction.

Laser-treated Specimens

After review of the tissues, it was determined that the soft tissue responses at the various exposure levels were similar, whether or not a restoration had been placed. Therefore, all laser-treated soft tissues were combined to provide *two areas in each animal* for each exposure time (10 seconds, 20 seconds, or 30 seconds). This yielded *12 soft tissue samples for each of the time periods,* 24 hours, 72 hours, or 5 days (Table 2).

24 Hours

Tissues were examined at 24 hours to determine the acute, immediate tissue reactions. At the recommended exposure level (10 seconds), no soft tissue alterations were noted in specimens removed after 24 hours. However, tissues exposed to twice the recommended laser exposure level (20 seconds) exhibited some slight connective tissue alterations in two of the four specimens. Those tissues exposed to three times the recommended exposure levels (30 seconds) exhibited notable connective tissue disruption in all specimens. The tissue changes ranged from slight inflammation (Fig. 3 and 4) with chronic inflammatory cells noted and a minor amount of connective tissue coagulation, to massive connective tissue coagulation and desiccation (Table 3).

72 Hours

Tissues examined at 72 hours showed the intermediate effects of laser energy. The soft tissue samples revealed a slight inflammatory infiltrate in all those areas exposed for 10 seconds. More severe alterations were present in all tissues after 20 seconds and 30 seconds of exposure. These changes were more extensive than those seen in the 24-hour specimens and ranged from disruption of the connective tissues to coagulation and necrosis of the underlying connective tissue (Table 4).

120 Hours (5 days)

The tissues exposed for 10 seconds were within normal limits. None of the alterations previously described were seen. Three of the four tissues exposed to 20 seconds of Argon laser still exhibited some tissue changes. Those exposed for 30 seconds all continued to exhibit notable tissue disruption (Table 5).

DISCUSSION

Although all tissues exposed to Argon laser responded with nonspecific disruption and necro-

TABLE 2. Restoration/No Restoration*

A. Restoration with laser polymerization		
Tooth #	Exposure tooth[a]	Soft tissue
R-M_1	15 sec	10 sec
R-P_4	25 sec	20 sec
R-P_3	35 sec	30 sec
B. Laser irradiation only (no Restoration)		
Tooth #	Exposure tooth	Soft tissue
L-M_1	10 sec	10 sec
L-P_4	20 sec	20 sec
L-P_3	30 sec	30 sec

*Restorations were placed in the indicated teeth of the right mandible. The corresponding teeth of the left mandible were not restored. Teeth of both left and right mandible and adjacent soft tissues received the same exposure: either 10 seconds, 20 seconds, or 30 seconds.
[a]An additional priming exposure of 5 sec was used to activate the bonding agent.

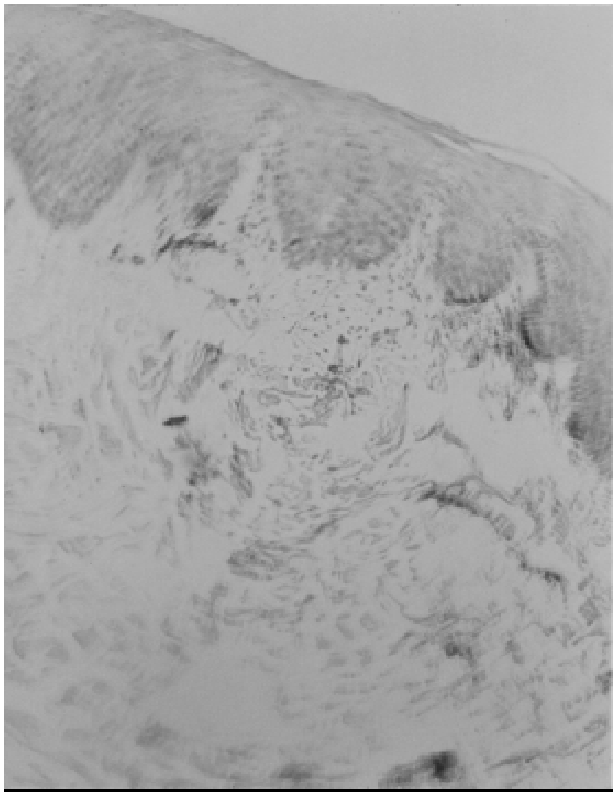

Fig. 3. Photomicrograph (Hematoxylin and Eosin X100) showing slight inflammatory cell infiltrate at the mucogingival junction and slight connective tissue coagulation representing the minimum changes exhibited at the lowest exposure level, 10 seconds.

sis of superficial tissues, those exposed to the recommended level (10 seconds) had minimal tissue damage and were found to be within normal limits when examined at 120 hours (Table 6). Those

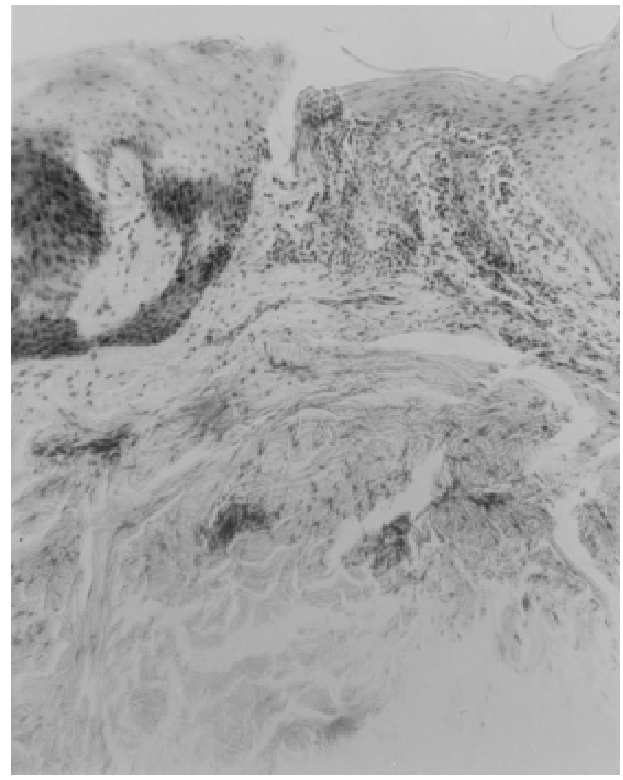

Fig. 4. Photomicrograph (Hematoxylin and Eosin X100) after a 30 second exposure at 72 hours postexposure, showing epithelial destruction, superficial connective tissue coagulation, and a chronic inflammatory cell infiltrate.

TABLE 3. Laser-exposed Tissues: Results at 24 Hours*

Tissue exposure	# of tissues	Result
10 sec	4	No alteration
20 sec	4	Scant C.T. disruption
30 sec	4	C.T. disruption

*After 24 hours the soft-tissue changes were as noted. They ranged from slight inflammatory infiltrate with chronic inflammatory cells to massive connective tissue coagulation.

exposed to increased amounts of laser energy (20 seconds and 30 seconds) had relatively greater tissue damage, and most still gave evidence of disruption at 120 hours (Table 6). As noted in the results, tissue changes were not seen in the control tissues exposed to Visible Light Cure, either associated with a restored or an unrestored tooth. Nor were the tissue changes seen in the untreated controls. Therefore, the tissue alterations examined are due to the laser irradiation.

The overlying parakeratinized epithelium was not extensively damaged by the laser exposure; however, the underlying connective tissue

TABLE 4. Laser-exposed Tissues: Results at 72 Hours*

Tissue exposure	# of tissues	Result
10 sec	4	Slight inflammation
20 sec	4	Coagulation of C.T. with inflammation
30 sec	4	Coagulation of C.T.

*The changes at 72 hours were more extensive and ranged from slight inflammation and disruption of the connective tissue to coagulation and necrosis of the connective tissue.

TABLE 5. Laser-exposed Tissues: Results at 5 Days*

Tissue exposure	# of tissues	Result
10 sec	4	No change (WNL)
20 sec	4	C.T. coagulation and fragmentation
30 sec	4	C.T. necrosis

*At 5 days, tissues exposed to the normal therapeutic dose (10 seconds) were again within normal limits. One of those exposed for 20 seconds was also within normal limits. The remaining tissues exhibited residual connective tissue coagulation, fragmentation and necrosis.

TABLE 6. Soft Tissue Damage Noted

Exposure	24 Hours	27 Hours	120 Hours
10 sec	0/4	4/4	0/4
20 sec	2/4	4/4	3/4
30 sec	4/4	4/4	4/4

*Compilation of tissue changes seen: at 5 days postexposure, five tissues had reverted to normal but eight still had evidence of injury.

was extensively hylanized and desiccated to the point of providing massive artifactual tearing of the tissue at the point of laser exposure. The dosages used were of very low energy levels, lower than that reported to produce histologic changes in orthokeratinized skin. Because the tissue subjacent to the parakeratinized epithelium was so extensively damaged, it appears that the reflection and subsequent loss of energy absorption reported in orthokeratinized epithelium is averted [8]. The laser energy appears freely to penetrate oral epithelium, causing greater damage to the underlying connective tissue than would be expected at these low energy levels. This coincides with the fact that in excess of 70% of the laser energy may be lost at the surface of keratinized tissue [9]. The parakeratinized epithelium exposed in this study allows a far greater amount of energy to penetrate the subjacent connective tissue where most of the tissue alterations were seen. The oral connective tissue is extremely well vascularized and, since Argon laser has an affinity for vascular tissue, the extent of connective tissue damage is not unexpected [5].

Particularly notable was the scant amount of inflammatory cell infiltrate seen in the histologic sections. Except in those sections with the greatest exposure, there were a minimal number of cells seen, even then they were all mononuclear cells. No evidence of an acute inflammatory cell infiltrate was present, which correlates with the findings by other authors [10].

Previous reports have shown that Argon laser exposure produces the most notable damage at 48 hours and that this may be due to increased transmission to the adjacent tissue producing lateral thermal destruction [7,11]. Tissues in this study also showed increased changes in the intermediate time period. Even those minimally exposed (10 seconds) had changes evident at 72 hours when no damage had been present at the initial time period of 24 hours. The tissues most exposed (30 seconds) had increased detrimental effects evident in tissues removed at the intermediate time period.

The damage incurred at 20 seconds of exposure was most noticeable at 72 hours. When the areas were examined at 120 hours, one of the four tissue samples was within normal limits. It appears that some resolution is possible within the 5-day time span. All of the tissue areas exposed for 30 seconds still exhibited complete disruption after 5 days.

Soft tissues around teeth both with restoration and without restoration were examined, since some alteration in these tissues may have occurred as part of the placement procedure regardless of the curring technique used. However, examination of the specimens revealed no obvious tissue differences whether or not a restoration had been placed. Therefore, it is evident that the alteration seen in the soft tissue are due to the Argon laser exposure rather than to mechanical manipulation.

Another aspect to consider is the difference in Argon laser reaction in pigmented tissues [9]. Resonant absorption occurs in pigmented cells, which selectively absorb at these wavelengths (488/514NM). Therefore, as some of our animals had pigmented gingiva, additional alterations were present including occasional epithelial disruption with ulceration.

During use as a polymerization instrument, Argon laser would never intentionally be applied to the adjacent soft tissues. However, these findings indicate that although some soft tissue dam-

age may occur at the recommended exposure level (10 seconds), lasting damage will not be seen. Other studies are being planned to investigate further the laser reactions in pigmented gingiva, in pulp, and in other associated oral tissues. A longer follow-up will allow investigation of the possibility of incomplete healing or residual damage.

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